The result of the experiments made is, therefore, very definite, and shows that the blood-serum of an immune calf contains something which neutralizes the specific virulence of vaccine virus, either bovine or from humanized lymph—crust from the arm of a child.

It is my intention to follow up this line of investigation, to endeavor to isolate this antitoxine of vaccinia, and to test the question of its possible specific action in neutralizing the smallpox virus in infected individuals, either before or after the development of the disease.

LOCAL ANÆSTHESIA AS A GUIDE IN THE DIAGNOSIS OF LESIONS OF THE LOWER SPINAL CORD.[1]

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Every fact which bears upon the exact localization of disease in the nervous system is entitled to most careful study, for the experience of the past ten years has demonstrated that the surgical treatment of nervous diseases—a treatment more radical, and sometimes attended by far greater success than medical treatment—must rest upon accurate diagnosis.

The study of motor disturbances, both of the form of paralysis and of the form of variations in reflex activity, has made it possible to determine the situation of lesions in the spinal cord with some exactness. The level of the cord at which the various reflex acts are performed is quite well known. The extent through various segments of the cord of those groups of cells which govern various muscles is also settled.[2]

But the spinal cord has sensory as well as motor functions, and it is only within a short time that the disturbances in sensation occurring in spinal lesions have been utilized for purposes of diagnosis.

This is partly due to the fact that the examination of sensory defects is tedious, and the results may be quite uncertain unless great care is taken in mapping out upon a convenient diagram the areas of anæsthesia. It may be partly due to the fact that the method of examination is often imperfect. For in examining for sensory defects it is always to be remembered that the test involves a contrast between two sensations, a process of comparison in the mind of one perception with another, and unless the two perceptions are presented simultaneously, such comparison is apt to be imperfect. It is not enough to ask a patient whether he feels the touch of the cotton-wool, or the tip of the finger, or the warm or cold test-tube, or the prick of the needle, or the sting of a faradic brush; nor is it sufficient to touch two parts successively, and demand that he compare a sensation with a memory. He should always be tested upon two surfaces simultaneously, and asked if any difference is felt between the sensations. The necessity of this is evident when it is considered, yet observation has convinced me that it is not the method commonly employed. In spinal affections we always have the face as a convenient surface with which to make comparisons; and in the diseases of the lower region of the cord, to which attention is directed in this paper, we have the arms and trunk as a standard of normal feeling.

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[1] Read before the Association of American Physicians in Washington, May 25, 1892.

[2] See "Syringomyelia," The American Journal of the Medical Sciences, May, 1888.

It has been my good fortune during the past few years to observe a number of patients suffering from disease in or about the lower half of the spinal cord, who have presented very interesting disturbances of sensation. In the medical journals and works I have found a few cases of more or less similar character—some of them with post-mortem records.

I hope to show from their study that a limited area of anæsthesia is produced by a limited lesion in the spinal cord; that as the lesion ascends the cord from its lowest limit upward, the area of anæsthesia extends in a definite manner upon the surface of the body; and that the situation and shape of the area of anæsthesia are positive indications of the level of the lesion in the spinal cord.

These facts are not wholly new. It has long been known that in transverse dorsal myelitis, the band of anæsthesia about the body corresponded to the level of the lesion. But there are many cases of myelitis of the cervical and lumbar regions of the cord in which the exact distribution of anæsthesia has been overlooked. This is especially true of lumbar and sacral myelitis, which is the form most frequently met with, and which alone will be considered in this paper. The situation of the anæsthesia in lesion of the lower part of the cord is such as to escape attention unless searched for; and as the patients lie in bed upon their backs, and sensation is often preserved in the front of the legs and thighs, the examiner, being content with the investigation of these parts, is misled into supposing that there is no anæsthesia.

The exact area of anæsthesia in lumbo-sacral myelitis has been investigated by various clinical observers, and isolated cases have been recorded.[1] Thus Westphal, Rosenthal, Huber, Bernhardt, Oppenheim, and Eulenberg, in Germany, Ross and Thorburn, in England, and Osler, Mills, Herter, and Church, in this country, have recorded cases. French observers who have made careful study of hysterical anæsthesia have appeared to overlook this subject. The only effort to bring the recorded cases together and make deductions from them has been made by Thorburn in his work upon the *Surgery of the Spinal Cord.* Since that work was written several important cases have been published with autopsies. And as I have had the opportunity of seeing six cases in which limited areas of anæsthesia aided the localization of the lesion, it has seemed proper to record these cases and review the subject carefully.

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[1] Westphal: Charité Annalen, 1882, i. 421. Rosenthal: Wiener med. Presse, 1887, xxix, 670. Huber: Wiener med. Wochenschr., 1888, xxxviii. 1310. Bernhardt: Berl. klin. Wochenschr., 1888, xxv. 637. Oppenheim: Arch. f. Psych., 1889, xx. 298. Eulenberg: Zeitschr. f. klin. Med., 1890, xviii. 547. Ross: Brain, 1887, x. 333. Thorburn: Brain, 1887, x. 381. Osler: Medical News, December 15, 1888. Mills: Medical News. March 1, 1890. Herter: New York Medical Journal, August 22, 1891. Church: THE AMERICAN JOURNAL OF THE MEDICAL SCIENCES, April, 1892.

The cases of special importance will first be described, and the conclusions from their study will then be considered.

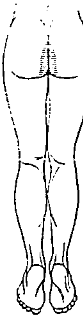
CASE I. (Starr.) *Injury; sphincteric paralysis; limited anæsthesia; lower sacral lesion.*—The first case is that of an engineer, J. L., who met with an injury in July, 1891, his engine falling some thirty feet, while he remained in a sitting posture in it, and thus received the blow upon his lumbar region, where there still remains the evidence of injury in a slight protrusion of the first and second lumbar spinous processes, with some induration about them. He found himself unable to rise after the fall, felt much pain in the lower sacral region and at the upper lumbar vertebræ, and at once lost control of his bladder and rectum. For five weeks after the injury he lay in his bed, though able to move his legs freely; and after this he got up and has walked, and now (March, 1892) walks perfectly well. The only evidence at any time of motor trouble was a twitching of the muscles of the outer side of the legs below the knees, which persisted for several months after the injury, but has now passed away. According to his statement, this twitching must have been limited to the peronei in both legs. For four months after the injury he was obliged to use a catheter and to wash out his rectum. At present (March, 1892) his sphincters are not under perfect control, his urine flowing as soon as his bladder is moderately full, though it never dribbles away, and his rectum occasionally emptying itself suddenly. He is however, slowly regaining control of both organs, but there is little resistance at the anus, and he wears a napkin. From the day of the accident to the present time he has felt a numb sensation in the lower sacral region, in the perineum, and back of the scrotum in the area shown in the diagram (Fig. 1). He is an intelligent man, and is quite certain that the sensory disturbances in this area have not varied, having been tested by several physicians since the injury. An examination shows a total loss of temperature- and pain-senses in the area marked out, and a marked diminution, but no loss, of tactile sense. The diminished tactile sense extends over the entire scrotum and posterior half of the penis, and is possibly the reason for an imperfect power of connection of which he complains, his sensations being slight, the flow of semen premature, and erection too short in duration, although he has impregnated his wife since the accident. There is no trace of anæsthesia of any kind elsewhere in his body. His knee-jerks are normal. The gluteal reflexes are absent. No ankle clonus. The complete absence of any symptoms of traumatic neurosis is interesting, and also the gradual improvement in the motor, reflex, and automatic symptoms, the only permanent symptom being the total anæsthesia to temperature and pain and the partial anæsthesia to touch in the limited area.

The diagnosis was a hemorrhage in the lowest part of the spinal cord.

CASE II. (Rosenthal.) *Sphincteric paralysis; limited anæsthesia; sacral lesion.*—Female, aged thirty-six years. After exposure to cold and fatigue, felt some pain in urinating and numbness in her buttocks. Four days later the pain and numbness had increased, she began to have retention of urine, and lost control of her rectum. Seven months later, when seen by Rosenthal, she was found to have anæsthesia to all sensation in the area shown in Fig. 2, including the genitals up to the mons veneris; she had no control of the bladder, which emptied itself spontaneously every two hours; she had no control of the rectum, and could move the bowels by enema only—but was perfectly able to walk. This condition had persisted for four years when the case was reported.—*Wiener med. Presse*, xxix. 670.

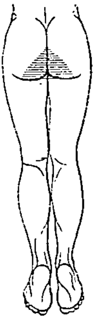
Rosenthal ascribed the condition to a hemorrhage, with destruction of the lowest part of the spinal cord.

FIG. 1.



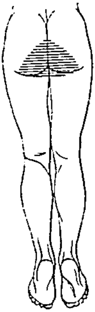
Starr.

FIG. 2.



Rosenthal.

FIG. 3.



Westphal.

CASE III. (Westphal.) *Sphincteric paralysis; limited anæsthesia. Autopsy—gumma of cauda equina.*—Female, aged thirty-six years; began to suffer in September, 1874, from pain in the legs and fatigue on exertion, with some numbness from the feet to the knees and a disagreeable sensation of pressure in the rectum. Soon afterward she lost control of the rectum, and began to have retention of urine. Then anæsthesia developed in the genitals, perineum, anus, and buttocks. When examined by Westphal, in December, 1874, she was found to be completely anæsthetic to touch, temperature, pain, and electricity in the area shown in Fig. 3, and also in the perineum, vagina, and over the external genitals up to the mons; there was a large bedsore in the anæsthetic area over the sacrum; there was retention of urine, which had to be drawn by catheter, the passage of which she did not feel; there was inability to empty the rectum, except by mechanical means; the anal sphincter was not relaxed, but grasped the finger. Motion was in no way impaired. She died December 27th, and the autopsy showed caries of the sacrum, and a gummy mass with hemorrhagic exudation and connective tissue filling the entire spinal cavity within the sacrum as high as the first sacral nerve, and compressing slightly all the sacral nerves except the first. The spinal cord was normal.—*Charité Annalen*, i. 421.

The following case, reported by Kirchkoff (*Arch. f. Psych.*, x.), is of interest, since it includes a record of post-mortem appearances.

CASE IV. (Kirchkoff.) *Injury; sphincteric paralysis. Autopsy—compression of sacral cord.*—The patient, a man aged thirty years, fell from his horse upon his nates, and felt immediately severe pain in his sacral region, and was unable to rise. For three months he was confined to bed, suffering from pain in the back and retention of urine, which was followed by incontinence of urine and feces; then he was able to go about, though he walked slowly with legs rather separated. Sensation is said to have been retained when he was examined one and a half years after the injury. He gradually recovered entirely, except for the incontinence of urine and feces, which remained till his death one and a half years after the injury. The autopsy showed that a portion of the body of the first lumbar vertebra had been fractured and was compressing the lowest part of the spinal cord, involving the fifth, fourth, and third sacral segments without compressing the cauda equina, the nerves of which were found normal on microscopic examination.

It is not at all impossible that there may have been a limited area of anæsthesia in this case about the perineum which escaped notice, especially if, as in my first case, the sensations of pain and temperature were the only ones permanently lost. Lachmann, however, has recorded (*Arch. f. Psych.*, xiii. 50) a case of tumor of the conus medullaris and filum terminale in which loss of sphincter control was present without any anæsthesia.

CASE V. (Starr.) *Spontaneous hemorrhage into the sacral cord; sphincteric paralysis; limited anæsthesia.*—My second case is that of a woman, R. K., aged twenty-eight years, who was seen with Dr. F. J. Quinlan in October, 1889, and in February, 1890, and whose condition since the last date has been stationary. She was perfectly well until May, 1889, when, after a day of fatigue, she was suddenly seized with severe pain in the sacral region and in the back of both thighs, with retention of urine and feces, and a sensation of numbness over the lower sacral region, in the perineum and vagina. She did not lose her power, and has been always perfectly able to walk. When examined in October, 1889, she was suffering much from pain in the lower part of the sacrum and along the back of the left thigh in the area shown on the diagram (Fig. 4). She had total anæsthesia to touch, temperature, and pain over the area shown, and this included the perineum and anus, and the entire genitalia, excepting the upper part of the labia outside the vulva. Her urine had to be drawn by catheter; her bowels were moved with great difficulty and with much pain. The sphincter ani contracted on the finger. Her knee-jerks were exaggerated. Plantar and gluteal reflexes were normal. There was no tendency to bedsores, no tenderness of the back to pressure or to heat, no girdle sensation, and no anæsthesia of the legs or thighs.

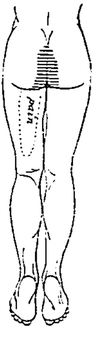
When examined again in February, 1890, she was still perfectly able to walk, but after long standing felt weak in the left leg, and found herself tired more easily than formerly. The leg was not at all atrophied, nor were the muscles paralyzed, but the response to faradism was diminished in the peroneal muscle as 60 to 45. Her bladder and rectum were still beyond her control, and were emptied by catheter and injections. She still had some pain in the back and in the left leg constantly, but recently had suffered more from pain in left ilio-inguinal region. There was no girdle sensation, spinal tenderness or bedsore. The area of total anæsthesia had increased, and now involved the area shown in Fig. 5. A slight degree of impairment of tactile sense was also found on the outer side of the left leg below the knee, and this could be traced from the anæsthetic region downward along a narrow space on the back of the thigh, indicated by the lighter shading in the figure. The original area of anæsthesia has therefore extended. The extension has been into the former area of pain, and the present area of pain has shifted from the back of the left thigh to the left groin.

It is also of interest to find that as the lesion has advanced upward and at last begun to invade the motor part of the spinal cord, it is in the peronei that the first sign of weakness—the diminution of the faradic contractility—has begun, thus confirming the statement of Leyden that these muscles are controlled by the lowest group of cells in the spinal cord (*Klinik der Ruckenmarkskr.*, i. 41).

Here, again, as in the first case, it is to be noticed that the areas of tactile disturbance and temperature and pain disturbance do not at present exactly coincide, and this seems to support the original diagnosis of a hemorrhage into the conus medullaris and lower sacral segments of the spinal cord. Such a hemorrhage might set up a chronic meningo-myelitis, and as this advanced slowly upward the symptoms would increase as they have done.

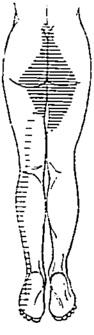
Such was the lesion in the following case of Oppenheim, which is reported with autopsy, and in which it will be noted that the final area of anæsthesia coincides quite closely with that in this case of my own.

FIG. 4.



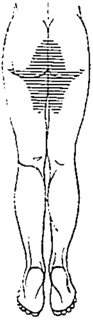
Starr.

FIG. 5.



Starr.

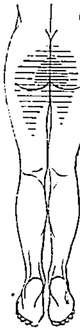
FIG. 6.



Oppenheim.

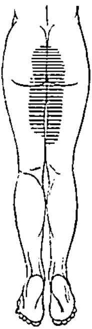
CASE VI. (Oppenheim.) *Injury; sphincteric paralysis, limited anæsthesia. Autopsy—hemorrhagic sacral myelitis.*—The patient fell upon his buttocks and immediately lost control of his legs, and felt a numbness in them. His urine was retained. He was taken to Charité Hospital, and observed there until his death, three months later. The permanent symptoms were: loss of Achillis tendon reflex, loss of control of the bladder and rectum, loss of power of erection, loss of sensation, especially for painful sensations in the area indicated in Fig. 6, which included "the entire vicinity of the anus, the gluteal, perineal-scrotal region and the penis, and a narrow area reaching downward on the posterior inner surface of the thighs, and upward over the lower half of the sacrum." It is to be noticed that the loss of the pain-sense was more complete than that of tactile sense. The autopsy showed a fracture of the first lumbar vertebra and a crushing of the posterior part of the conus medullaris and disintegration of the lower two-thirds of the sacral portion of the cord. "In the lowest portion the posterior half of the spinal cord seemed entirely destroyed, as if it had been cut off by an incision through the posterior columns 1–2 mm. behind the posterior commissure. The boundary is, however, by no means a regular one, but was encroached upon by a thick, very vascular tissue full of round cells, a part of the pia mater infiltrated with granular cells, small and large collections of blood-cells and new capillary vessels. The anterior part of the cord was also altered, being infiltrated with small round cells, while the nerve-cells were in part disintegrated. It was evidently a traumatic myelitis with hæmato-myelia." The only lesion in the lumbar enlargement was the ascending degeneration in the posterior columns. The nerve-roots coming from the lowest part of the cord were involved in the lesion, being matted together in the thickened pia, but a microscopic examination showed that the nerve-roots which issued from the two upper sacral and lumbar portions of the cord were not affected, although they had to pass through the narrowed canal.—*Arch. f. Psych.*, xx. 298.

FIG. 7.



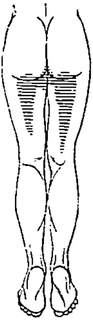
Huber.

FIG. 8.



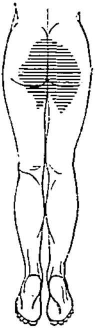
Bernhardt.

FIG. 9.



Osler.

FIG. 10.



Mills.

Cases almost identical in character are reported by Huber,[1] Bernhardt,[2] Osler,[3] and Mills,[4] without autopsies. The area of limited anæsthesia is shown in the accompanying figures. (Figs. 7, 8, 9, and 10.)

In the following case, reported by Herter, which was seen by me at his request, the area of anæsthesia was slightly more extensive. The case was completed by a pathological examination.

CASE VII. (Herter.) *Injury; paraplegia, sphincter paralysis, limited anæsthesia. Autopsy—hemorrhage in spinal cord.* Male, aged thirty years, was crushed by a heavy weight on August 26, 1890, and immediately paralyzed in the legs and had involuntary passage of urine and feces. Five weeks later, when the effects of the shock had disappeared, the paralysis of the feet and legs remained with atrophy and reaction of degeneration below the knees, but the thighs could be moved; the bladder and rectum were totally paralyzed, neither sphincter offering any opposition to pressure; sexual power was lost; a bedsore had formed over the sacrum, and anæsthesia was present in the area shown in the diagram (Fig. 11) and included the perineum and genitals. The darker area over the buttocks was totally anæsthetic to touch, temperature, and pain; the lighter area was partially anæsthetic. The soles and dorsal surfaces of the feet were quite anæsthetic and analgesic. The patient improved during the following five months so that he was able to walk, but the anæsthesia remained the same. I saw the patient, with Drs. McCosh and Herter, in January, 1891. From the beginning there had been a prominence of the first lumbar spine, and on January 27, 1891, the operation was undertaken, and the spines and laminæ of the first, second, and third lumbar vertebræ were removed, and the cauda equina was exposed. Nothing could be found at the operation. The patient died forty-eight hours later.

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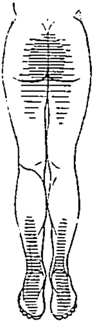
[1] Wien. med. Wochenschr., 1888.

[2] Berl. klin. Wochenschr., 1888.

[3] Medical News, 1888.

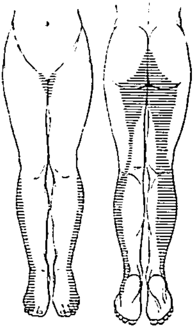
[4] Ibid., 1890.

FIG. 11.



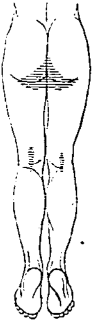
Herter.

FIG. 12.



Eulenberg.

FIG. 13.



Eulenberg.

The autopsy showed the existence of an old hemorrhage and a mass of inflammatory material in the cauda equina and involving the lower three-quarters of an inch of the spinal cord. The nerve-fibres of the most centrally situated strands of the cauda were completely degenerated. The anterior strands also contained degenerated nerve-fibres. The lateral bundles escaped. The conus medullaris was destroyed. The lesion did not reach as high as the fourth lumbar roots, which were normal.—HERTER, *N. Y. Med. Journ.*, Aug. 22, 1891.

In the following case, recorded by Eulenburg, the hemorrhage was spontaneous and not traumatic in origin, and the subsidence of the symptoms indicated a gradual absorption of the clot.

CASE VIII. (Eulenberg.) *Sphincter paralysis, limited anæsthesia.*—Female, aged thirty-six years; had suffered occasionally for five years from endometritis and pains in back and legs, and was suddenly seized at Christmas, 1887, with severe pain in the back and fell to the ground. For several weeks after this she was in bed, and on getting up limped in walking. In April, 1888, she had another attack of pain in the sacrum, and then developed paralysis of the bladder, with retention of urine and loss of sensation below the waist. Three months later the rectum became paralyzed, being emptied with enema with difficulty, though occasionally an involuntary movement occurred. She was first examined by Eulenberg in February, 1890. She then had pain in the back and in the bladder, with tenderness over the sacro-lumbar articulation. There was anæsthesia in the genitals, perineum, anus, and over the region shown in Fig. 12, the dark shading indicating a total anæsthesia. The gait was slow and uncertain, stepping up being difficult, as were also flexion of the knees and plantar flexion of the feet, the left calf and thigh being weaker than the right and slightly atrophied. Knee-jerks were normal. Achillis reflex lost, and plantar reflex imperfect. No gluteal reflex. All muscles acted to faradism, but it required a stronger current to contract the peronei and ante-tibials than the post-tibials. The bladder was usually emptied by catheter, but spontaneous evacuation occurred frequently. The rectum was beyond control and the sphincter ani was weak. Menses were regular.

Under treatment by strychnine and static electricity there was some improvement, and at the end of four and a half months the anæsthesia was diminished to the limits shown in Fig. 13, the genitals being still affected, this being the region originally of total anæsthesia; there was still imperfect control of bladder and rectum, but the power of the limbs seemed to have returned.—EULENBERG, *Zeitschr. f. klin. Med.*, xviii. 547.

These cases seem sufficient to demonstrate that small lesions in the sacral region of the spinal cord may produce limited zones of anæsthesia on the back, thighs, and outer side of the legs, and that the extent and shape of the anæsthesia vary with the extent or situation of the spinal lesion.

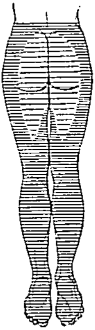
In contrast to these cases the following is of special interest, as it shows that in limited myelitis of the lumbar region the sensory functions of the sacral segments may be preserved and impulses may pass the diseased area of the cord and thus reach the brain.

CASE IX. (Starr.) *Central myelitis of the lumbar region, limited anæsthesia; paraplegia.*—The patient, a young married lady, after suffering for some months from backache, was suddenly seized, in May, 1891, with severe pain in the lower dorsal and upper lumbar region, which was much increased by motion and by lying down, and which was soon followed by pain down the left leg in the sciatic distribution. In August, 1891, a swelling appeared at the third lumbar vertebra which gradually disappeared in a month. Its nature is undetermined. On the 8th of August she suddenly lost sensation to heat and to pain in the left leg. On the 16th of September she lost power of motion in the right leg except in the foot. In October the left leg became paralyzed and sensations of touch were also lost. Finally, in November, the right leg became anæsthetic. Early in the disease retention of urine and obstinate constipation appeared and have continued, so that catheterization is necessary. During November the line of anæsthesia advanced slowly upward from the groins to a point half-way to the umbilicus, where it remains.

When she was seen in December, 1891, in consultation with Dr. J. G. F. Holston, there was total paralysis of motion below the waist, including back and pelvis, together with atrophy more marked above than below the knees. All reflex action was abolished. The muscles all reacted to a very strong faradic current, the reaction below the knees being more prompt than above, and there being normal galvanic response below the knees, and equal response to cathode and anode above the knees. The limbs were flaccid—cool, without rigidity—and the occurrence of bedsores, to which there is a tendency in the back, was prevented by frequent change of position. The loss of sensation to touch, temperature, pain, and muscular sense was absolute below the middle of the lower abdomen and first lumbar spine, but on the back, in a saddle-shaped area of the surface shown in the diagram, Fig. 14, these sensations were not lost but only slightly diminished. This area was determined independently by Dr. Holston and myself at different times. This area of sensibility included the anus, perineum, and vagina. The bladder was under no voluntary control, urine being retained till drawn or passed in a stream when the bladder became full, and slight cystitis being present unless the bladder was constantly washed out with borax solution. The sphincter ani offered resistance to the finger. The evacuation of the rectum was difficult with enema, and always painful.

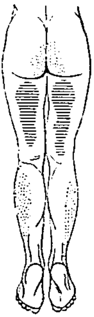
Here there was evidence of a total loss of all the functions of the lumbar segments of the cord, including the first, and of the *integrity of the sacral segments*. Sensory impulses were received in the latter, and made their way upward through the affected lumbar region—a fact which aids the diagnosis, inasmuch as it demonstrates that the myelitis in this region does not affect the posterior columns of the cord at that part, but must be confined to its gray matter and lateral portions.

FIG. 14.



Starr.

FIG. 15.



Starr.

The case is stationary at present (April, 1892). In regard to its diagnosis, it is quite possible to exclude a tumor compressing the cauda equina because of the order of appearance of the symptoms: 1, anæsthesia in left leg; 2, paralysis in right leg; 3, anæsthesia in right leg; 4, paralysis of left leg, an order implying the invasion of the spinal cord from its right half over toward its left half, producing at one stage a true picture of a Brown-Séquard paralysis. The possibility of an intra-spinal tumor or gliomatosis cannot be excluded, and it is very likely the cause of the destructive myelitis present. An operation has, however, been advised against, as it seems improbable that an intra-spinal tumor could be removed with any prospect of recovery, the necessary destruction of the cord during the operation being liable to leave as serious results as the tumor is now producing. And the fact that sensory impressions pass up the cord at the seat of lesion, indicates that the tumor does not lie on the posterior surface where it might be most easily reached in an operation.

CASE X. (Starr.) *Syphilis of the spinal cord; sphincteric paralysis, limited anæsthesia and hyperæsthesia.*—Male, aged twenty-two years (Vanderbilt Clinic, No. 4711), had syphilis in January, 1891, with well-marked secondary symptoms from which he recovered. In November, 1891, he felt some weakness and numbness of the legs. In December, 1891, after dancing all night, found that he could not pass his water and had to have it drawn. At the same time noticed an increased numbness of his legs and felt weak in walking. Felt as if he were walking on a carpet. These symptoms increased gradually and have continued till the time he was first seen (February 11, 1892). Examination showed inability to control his urine, which at times flowed away when the bladder was full, and at all times was retained; constipation, a slight weakness of the legs, exaggerated reflexes and tendency to spastic gait, and a condition of hyperæsthesia to touch, temperature, and pain in area dotted in the figure (Fig. 15), over buttocks and on the outer calf of each leg and an area of anæsthesia of slight degree over the back of each thigh between the hyperæsthetic areas.

Under anti-syphilitic treatment and rest he recovered entirely in the course of a month.

The presence of a zone of hyperæsthesia around the body in transverse dorsal myelitis is often recognized. It is interesting to find areas of hyperæsthesia in this case, one corresponding to the area of anæsthesia from lower sacral lesions, one corresponding to the area of anæsthesia in lower lumbar regions, with a zone of anæsthesia between them.

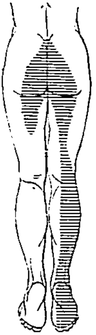
The diagnosis of syphilis of the cord may be thought indefinite, and it is not impossible that at the time of sudden increase of his symptoms he may have had a slight hemorrhage into the spinal membranes over the sacral cord. But Erb has recently called attention to a peculiar association of symptoms which he considers characteristic of syphilis of the cord, and the patient's symptoms correspond closely to that condition. These symptoms are slight spastic gait and weakness, with increased reflexes but with very little muscular rigidity, interference with the bladder functions, and slight disturbances of sensation chiefly of the nature of paræsthesia in the legs. There are no symptoms above the waist. The onset is at first gradual, but the symptoms may increase suddenly, and under treatment may pass away. It will be seen that this case corresponds quite closely to this description.

CASE XI. (Starr and Lloyd.) *Injury; compression of cauda equina; localized paralysis; sphincteric paralysis; limited anæsthesia; operation; recovery.*—Male; was struck, in April, 1889, on the back while in a kneeling posture, and sustained a fracture of the lumbar vertebræ. He was immediately paralyzed in his right leg; had loss of bladder and rectal control, and a considerable degree of anæsthesia whose area is uncertain. A year later he was seen by me with Dr. Samuel Lloyd.[1] There remained a deformity of the lumbar region most marked at the third lumbar vertebra. There was total paralysis with atrophy and reaction of degeneration of the peronei, anterior and posterior tibial and glutei muscles of the right leg, and some weakness in the muscles of back of the thigh. There was diminished knee-jerk and no ankle clonus. There was loss of control of the bladder and rectum. There was anæsthesia in the area shown in the diagram (Fig. 16) to touch, temperature, and pain.

The diagnosis of compression of the cauda equina at the third lumbar level being made, Dr. Lloyd operated and removed the second and third lumbar spines and laminæ. The patient subsequently recovered very slowly but entirely from all his symptoms.

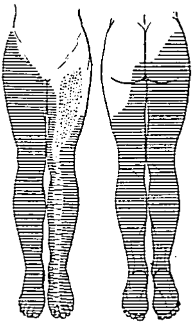
CASE XII. (Starr and McBurney.) *Injury; paraplegia, limited anæsthesia; operation; improvement.*—Male; suffered a fracture of the lumbar vertebræ in June, 1891, and was immediately paralyzed in both legs; lost control of his sphincters and had a loss of sensation in the legs. This anæsthesia, he said, was at first as high as the umbilicus and extended about the body, but within a month of the injury decreased slightly and had become permanently limited to the regions shown in Fig. 17. Bedsores developed soon. His condition had been stationary from August to October, when he was seen by me in Roosevelt Hospital at Dr. McBurney's request.

FIG. 16.



Starr and Lloyd.

FIG. 17.



Starr and McBurney.

I found a total paralysis with reaction of degeneration of all the muscles of the lower extremities, with atrophy, loss of reflexes and of mechanical excitability, the abdominal muscles, however, being under control, and abdominal reflex preserved; complete loss of sensation in the area shown in the figure, to touch, thermic and painful and electric irritation, and an area of marked hyperæsthesia on the left thigh in front over the region dotted. He had no idea of the position of his feet and legs in bed or when moved. He had no control over his bladder, and his rectum was evacuated with difficulty by enemata. He had a deep bedsore over the sacrum, and bedsores on the ankles were prevented with difficulty. The deformity at the first, second, and third lumbar vertebræ was well marked, the second lumbar spine being deflected an inch to the right.

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[1] Dr. Lloyd has reported the case in full in THE AMERICAN JOURNAL OF THE MEDICAL SCIENCES, July, 1891.

On October 31st Dr. McBurney removed the spines and laminæ of these vertebræ, revealing a fracture of the body of the second lumbar vertebra. The cauda equina was compressed between the body of the vertebra, which had been split off and projected backward, and the laminæ chiefly on the right side. When the cauda was entirely freed, pulsation returned in its vessels and in the dura. The dura was opened necessarily, and it was evident, from the appearance of the nerve-trunks, that they were degenerated. The wound healed promptly. The bedsores healed within three weeks of the operation. The control of his bladder and rectum had partially returned three months later, but no change in motion or sensation had been produced at the end of six months. He has suffered very greatly from pain in his legs ever since the operation.

The areas of anæsthesia and hyperæsthesia are interesting, and must have been due to compression of the entire cauda equina at the second lumbar nerve, being greater on the right side than on the left. It is evident that the bundles of the cauda lying on the surface on the left side were the only ones to escape.

Conclusions.—There are a number of interesting facts to be drawn from the study of the cases here collected.

First, *as to the location in the spinal cord of the centres of control of the bladder and rectum.* These centres appear to be uniformly affected together, and, therefore, must be adjacent to one another. The control of the sphincters is lost when the lesion involves the lower three sacral segments, and the centres probably lie in the lower two segments of the cord. This is proven by the autopsies in the cases of Kirchkoff, Westphal, Oppenheim, and Herter, and by the distribution of sensory symptoms in two of my cases, and in those of Rosenthal, Bernhardt, Eulenberg, Mills, and Huber. In these cases without autopsy the situation of the anæsthesia was such as to show that the lesion was limited to the two or three lower sacral segments, and in all the control of the sphincters was lost.

When these segments are destroyed, the sphincter of the rectum is relaxed and there is no opposition to the introduction of the finger into the anus. The entire rectum also loses its power of contraction so that it is only emptied by pressure from above or by artificial evacuation by means of enemata or excavation.

The sphincter of the bladder does not appear to be permanently relaxed when the cord is destroyed. At any rate a constant dribbling of the urine rarely, if ever, occurs. There is, however, a moderate incontinence, for as soon as a few ounces of urine collects in the bladder the pressure overcomes the slight resistance of the sphincter and the urine flows away. Hence a frequent emptying of the bladder without the knowledge of the patient takes place. There is rarely a sufficient resistance offered by the sphincter to cause a retention of urine and distention of the bladder when the lesion destroys the bladder centres. This is much more liable to occur when the lesion lies at a somewhat higher level in the upper sacral region and produces an irritation of the mechanism of the bladder. It seems to occur also when the lesion involves the cauda equina, producing pressure on the nerve roots. Since pressure upon a nerve elsewhere never produces tonic spasm, this effect must be of a reflex nature from compression of the sensory filaments.

If, in a case of paraplegia, the mechanism of the bladder and rectum is not interfered with—if these organs empty themselves naturally when full—in spite of or without the knowledge or control of the patient, it is a proof that the lesion has not destroyed the lower sacral segments of the spinal-cord. In such cases the exact area of anæsthesia should be carefully determined, for the information thus afforded may enable an exact diagnosis of the situation of the lesion and also of the actual extent on the cord to be determined. (See Case IX.)

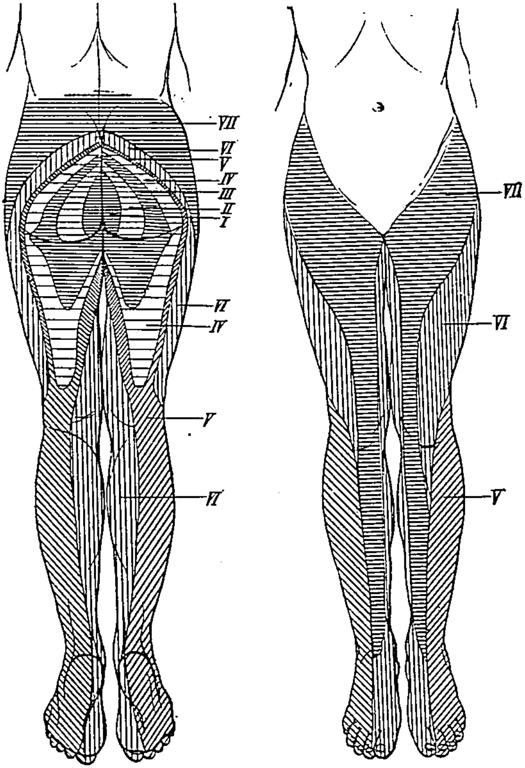
Secondly, *as to the distribution of anæsthesia in lesions of the lower part of the spinal cord.* This is shown in the diagrams (Fig. 18). These diagrams have been prepared by carefully comparing and superposing the charts of cases cited. In some cases the area of anæsthesia was strictly limited from the outset to a special region. In other cases the area of anæsthesia was large at the outset and gradually diminished in extent. In other cases still the area of anæsthesia was at first small and then increased. The three sets of cases, therefore, must agree in their results, in order to establish any conclusion as to the areas of anæsthesia produced by lesions at various levels. And if the cases here collected be carefully studied, it will be seen that they do thus agree. In the diagram of the back it is possible to outline seven concentric zones of anæsthesia, starting from the lowest part of the sulcus between the buttocks as a centre. These are numbered *I.* to *VII.* in the diagram.

I. The *first zone* is oval in shape, small in extent, and includes the perineum, the posterior part of the scrotum in males, the vagina in females; it also includes the mucous membrane of the rectum.

II. The *second zone* is heart-shaped—point up—and includes the entire scrotum and posterior surface of the penis and mucous membrane of the urethra in males—the entire genitals of the female, except the outer surface of the labia majora and the mons veneris.

These two zones can be separated clinically, for in some cases the smaller zone only has been anæsthetic. In the majority of cases the larger zone has been found anæsthetic. No autopsies are at hand to enable a distinction of lesion to be made between these two zones, but from the cases of Kirchkoff, Westphal, and Herter it is possible to affirm that the second zone is produced by a lesion involving the conus medullaris and the fifth and fourth sacral segments of the cord.

Fig. 18.



Areas of anæsthesia in lesions at various levels of the spinal cord from sacral v. to lumbar ii.

I. Sacral v.
II. Sacral iv.
III. Sacral iii.
IV. Sacral i.
V. Lumbar v.
VI. Lumbar iii.
VII. Lumbar ii.

III. The *third zone* is considerably larger, involving a greater surface of the buttocks and extending down the back of the thighs over a triangular area, point down. This has been named the "saddle-shaped area," coinciding about with the surface of the seat in contact with the saddle when riding. A zone of anæsthesia of this shape is due, as the autopsy in Oppenheim's case shows, to a lesion involving the fifth, fourth, and third sacral segments.

IV. The *fourth zone* is of a similar shape to the third, but more extensive, a greater surface on the back of the thighs being involved, and the anæsthesia extends in a band almost as low as the popliteal space. This area has been established clinically in several cases: there is as yet no autopsy to determine its lesion; but since the smaller zone is due to lesion at the third sacral segment, and the next larger zone is due to lesion in the fifth lumbar segment, it is allowable to conclude that this region corresponds with the second and first sacral segments.

In thus outlining four zones of the skin and assigning them to various segments of the sacral portion of the spinal cord, it is not my intention to lay down artificial boundaries or to affirm that all cases will exactly coincide. The lesions in the sacral cord are not limited exactly to one or two segments. The sacral cord is small in extent and lesions involve it to a greater or lesser degree, consequently these zones are not always symmetrical on the two sides of the body; the lesion being a little higher on one side of the cord than on the other, the zone of anæsthesia will be greater on one thigh than on the other, as in my first, fifth, and sixth cases, and in those of Herter, Mills, and Osler. I only wish to show that as the cord is invaded by disease from below upward, the area of the skin which becomes anæsthetic increases in extent, and that the shape of the area is characteristic; so that from the study of the area the extent of the lesion can be determined.

I also desire to call attention to the fact observed in my fourth case, that this area of the skin which corresponds to the sacral cord may remain sensitive in a lesion of the lumbar cord, when that lesion is so limited in extent as not to cut off impressions passing from the sacral cord to the brain. This affords an important evidence of the extent of a lesion in the lumbar cord. For if that lesion is transverse and involves all the columns, all sensation below its level will be lost, while, if the lesion is limited and does not affect the posterior columns of the cord through which impulses are passing up from the sacral region, the total anæsthesia will not include the parts of the skin related to the sacral cord.

Another conclusion of some importance is brought out from a study of these facts, viz., that in locomotor ataxia the lesion probably begins quite uniformly in the lumbar cord, and not in the sacral cord. In a review of about fifty cases of locomotor ataxia, of which I have careful records, I find that the lightning pains of the initial stage and the hyperæsthesia especially to cold, which accompany them, are rarely referred to the buttocks and seat. They are quite uniformly referred to the feet or to the legs below the knees or to the front and outer sides of the thighs. Pain in the rectum, it is true, may be felt. In one of my cases the initial paræsthesia and anæsthesia occurred in the rectum and perineum, the complaint for several months being of a feeling as if a large cannon-ball were lying on the perineum. But such localization of pain and paræsthesia is the exception. If, however, the lesion of locomotor ataxia began in the sacral portion of the cord, it is reasonable to suppose that the sensory symptoms would be referred to the region of the body corresponding to that part of the cord, and that bladder disturbance would be the first symptom. It seems, therefore, quite probable that the lesion in locomotor ataxia begins in the lumbar region—a fact to which the early loss of knee-jerk also points. This may have a bearing on the disputed question as to whether locomotor ataxia is a true "system disease."

The area of anæsthesia produced by lesions in the lumbar cord is also determined by these cases and shown in the diagram.

V. The *fifth zone* of anæsthesia is seen to include the first four zones and to extend down the back of the thigh through the popliteal space in a band, and then to descend the outer surface of the leg to the foot. In some cases it ends at the ankle, in others it involves the entire side of the foot, dorsum, and sole, and three and a half toes. Eulenberg's case, cases of Kahler[1] and Mills[2] not cited here, and my second and fifth cases demonstrate this distribution. When a lesion extends from the sacral into the lumbar cord the anæsthesia extends from the thigh down the outer side of the leg. This area then corresponds to the fifth lumbar segment of the cord.

VI. The *sixth zone* of anæsthesia is produced by a lesion of the third lumbar segment. When the third lumbar segment is diseased, the entire back of the thighs and legs is anæsthetic and the front of thighs is also anæsthetic, except over a funnel-shaped zone which extends from above downward, the narrow tube of the funnel reaching along the shin even to the foot. This distribution of anæsthesia is well shown in Fig. 17, where the sensitive area in the midst of the anæsthetic zone is well defined. This zone will probably be separated later into two separate parts corresponding to lesions of the fourth and third lumbar segments. There is not as yet a sufficient number of cases to warrant such a distinction. The exact limits of anæsthesia on the feet are still uncertain, and no more exact statement than that given is warranted. It is quite common to find the inner arch of the foot sensitive when the toes and heel and entire dorsum are anæsthetic, and it is probable that the higher the lesion the greater the anæsthesia on the foot.

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[1] Kahler: Prager med. Wochenschr., 1882.

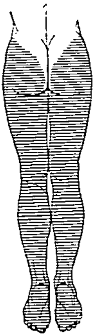
[2] Mills: Med. News, March 1, 1890, Case I.

VII. The last and *largest zone* of anæsthesia shown in the diagram is produced by a lesion of the four lower lumbar segments, that is, by destruction of all but the first lumbar segment of the cord. It will be noticed that the line of anæsthesia is much lower in front than behind, and that it follows the line of Poupart's ligament. It is only when the first lumbar segment of the cord is invaded that the abdominal wall becomes anæsthetic.

From this level upward the zone of anæsthesia extends around the body in a girdle, and there is no difficulty in locating the level of the lesion in the dorsal cord.

It is to be remembered that in all these lesions and areas of anæsthesia, the *anus, perineum, and the genitals are included in the insensitive region.* This is an important fact in the differentiation of cases of organic from functional paraplegia. It is also to be noticed that the shape of the area of anæsthesia in the back differs in organic and in functional cases.

Fig. 19.



Charcot.

Charcot[1] and his pupil, Souques,[2] have described the area of anæsthesia in hysterical paraplegia, in paraplegia produced by hypnotic suggestion, and in the analogous form of functional paraplegia occurring after railroad and other injuries, and included under the general name of traumatic neurosis or traumatic hysteria. Fig. 19 shows Charcot's diagram. He says: "In front the limit of anæsthesia is represented by a line which follows the fold of the groin on each side to the anterior spine of the ilium, *exclusive of the genital organs;* on the back the line follows the insertion of the muscles of the buttocks exclusive of a V-shaped area over the sacrum." It will be noticed how entirely different this area of anæsthesia is from that due to any of the organic lesions described here. And the escape of the genital organs in the functional cases is also a most important point of diagnosis between hysterical and organic paraplegia.

The study of the area of anæsthesia, therefore, not only enables us to reach a diagnosis of the level of the lesion; but it also enables us to separate organic cases of spinal-cord disease from functional and hysterical cases of a paraplegic kind.

Multiple neuritis is sometimes attended by anæsthesia of the extremities. The anæsthesia is limited, however, to the hands and feet, or if it extends up the limbs it is limited by a line drawn around the legs or forearms below the knees or elbows. It never resembles in its distribution the area of anæsthesia produced by lesions of the spinal cord.

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[1] Charcot: Œuvres complètes, iii. p. 448.

[2] Souques: Iconographie de la Salpêtrière, iv., 1891.

Nor can the anæsthesia from injury of special nerve trunks be confounded with that due to spinal disease, as a reference to any diagram of the distribution of the cutaneous nerves will show.

Thirdly, in cases of transverse dorsal myelitis with a zone of anæsthesia about the body, there is usually present a zone of hyperæsthesia just above the zone of anæsthesia. An analogous condition has been observed in cases of lumbar and sacral myelitis, as shown by three of my own cases (Cases V., X., XII.). But in these cases the region of hyperæsthesia was situated in the area of the surface corresponding to the segments of the cord just above the lesion. In Case V. the area of anæsthesia corresponded to a lesion of sacral V. and IV., the area of pain and hyperæsthesia corresponded to sacral III. As the disease extended, the zone formerly hyperæsthetic became anæsthetic. In Case X. the area of anæsthesia corresponded to a lesion of sacral III. and IV.; the hyperæsthesia corresponded to sacral IV. and V. below, and to sacral I. above. In Case XII. the area of anæsthesia corresponded to a lesion of lumbar III. and below it; while the area of hyperæsthesia on the left side corresponded to the funnel-shaped area belonging to lumbar II. Thus the study of the areas of hyperæsthesia confirms the conclusions reached from a study of the areas of anæsthesia.

It may be affirmed that in lesions of the lower cord an area of hyperæsthesia will be usually discovered in the part of the body corresponding to the segment of the cord just above the lesion. And when a lesion is extending, the area previously hyperæsthetic will become anæsthetic. If the diagram be consulted, some conclusion regarding the extent of any given case can be drawn from a study of the relative positions of the areas of hyperæsthesia and anæsthesia.

Lastly, a few words must be added regarding the differentiation of lesions of the spinal cord from those of the cauda equina. The study of the anæsthesia alone does not aid very greatly in the differentiation. A lesion of the sacral nerve roots produces an identical area of anæsthesia as a lesion of the sacral cord. It also produces a paralysis of the bladder and rectum. Thorburn[1] has proved that pressure exerted on the cauda equina affects the nerves in the middle of the cauda to a greater extent than those near the surface. "Those nerves which pass out lower down are, in the cauda, situated nearer the middle line than those which pass out above them, and hence they would appear to have more room to escape from pressure, and we might expect them to suffer less rather than more; but that the contrary is the case is an established fact, and we are able definitely to conclude that in a pressure lesion of the entire cauda equina those nerve roots which emerge lower down are more seriously injured than those above them." This conclusion has been proven by the autopsy in Herter's case, where the middle roots only were degenerated, though the pressure was exerted on all the roots at the level of the last lumbar nerve.

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[1] The Surgery of the Spinal Cord, p. 99.

There is no way, therefore, of determining by a study of the anæsthesia alone a pressure lesion on the cauda high up from a destructive lesion in the cord at its lowest extremity.

The diagnosis may, however, be made, first, from a study of the surgical indications, chiefly of the nature of deformity, the relation of the vertebræ to the segments of the cord being remembered. The cord ends at the first lumbar vertebra, hence any fracture below that is necessarily compressing the cauda equina. Secondly, the diagnosis may be made from a study of the paralysis which accompanies the anæsthesia. This paralysis is very slight in lower cord lesions, being confined to the peronei muscles when the lesion is at or below the second sacral segment; it involves the anterior tibial and posterior tibial muscles when all the sacral segments are involved, and only invades the movements of the hip-joint when the entire lumbar region of the cord is affected.[1] In cauda lesions, on the other hand, the pressure on the nerve roots is often sufficient to produce widespread paralysis when sensation is but slightly affected.

The cases in which autopsy or operation has revealed the nature of the lesion in diseases of the lower cord demonstrate, however, that a sharp differentiation between cauda equina and cord lesions is not often justifiable. In the four autopsies here cited, both cord and cauda were invaded by the lesion, which was a meningo-myelitis with hemorrhage, the result being a destruction of the lower cord and a matting together of the nerves of the cauda in a mass of inflammatory material. It seems, therefore, questionable whether, except in cases of fracture below the first lumbar vertebra with displacement of the vertebræ, any sharp line of distinction between cord and cauda lesions should be attempted. It is chiefly in the surgical cases that operative interference has been attempted, and here, as already stated, the surgical rather than the medical facts have been the surest guides to the operation.

There is one case on record, however, of successful operation for removal of a tumor compressing the cauda equina (Laquer, *Neurol. Centralbl.*, 1891, x. 193). In this case the extreme pain in the sacral region and the tenderness over the sacral region appear to have been the particular symptoms which guided the operator; the tumor was extra-dural, and the nervous symptoms were by no means such as to suggest a lesion of the spinal cord. In two other cases of tumor of the cauda equina—viz., those of Simon (*Arch. f. Psych.*, 1875) and Lachmann (*Ibid.*, 1882), the characteristic nervous symptoms of compression of the cauda were wholly wanting, and the cases were not diagnosticated during life.

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[1] For more precise statements see "Syringomyelia," THE AMERICAN JOURNAL OF THE MEDICAL SCIENCES, 1888.

Some writers upon the differential diagnosis between cord and cauda equina lesions have laid stress upon the fact that sensations of touch, temperature, and pain are not always equally destroyed, and have sought to conclude that this inequality of sensory disturbance was evidence of cord lesion as distinguished from cauda equina lesion. The cases here cited of Herter and Oppenheim, with autopsies, and two of my cases, in which the operation showed the lesion to be a compression of the cauda without lesion of the cord, prove that this point of differential diagnosis is not well taken. In both cord and cauda lesions the disturbance of tactile sense may be more or less extensive than that of the senses of pain and temperature.

It is evident from the facts here presented that a careful study of disturbances of sensation is a valuable aid in the diagnosis of the situation of lesions in the spinal cord and cauda equina. It is, however, to be remembered that anæsthesia is but one of a series of symptoms entering into that diagnosis, and the condition of reflexes, and the power, tone, and electric reactions of the muscles are not to be neglected in the examination of any case. It is only when *all* the signs of a local lesion coincide that the diagnosis is an absolute one.

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RADICAL CURE OF PSOAS ABSCESS.

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THE treatment of psoas abscess secondary to disease of the spine has from time immemorial been extremely unsatisfactory, and even at the present day many different practices are followed.

Deckers, writing in 1693, advised opening the abscess with a trocar; he left the canula in the cavity, stopped with a *cork*, and let out the pus at intervals. Benjamin Bell adopted a similar proceeding.

Liston advised opening the abscess when it pointed, and says: "In these cases the discharge is generally profuse, long continued, and attended with exhaustion and hectic, gradually but surely destroying the patient." "Opening the cavity and shutting it up again, however carefully conducted, is in almost every instance followed by alarming and hazardous results." Lawrence and Sir Astley Cooper, on the other hand,